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Patterns, Variability, and Predictors of Urinary Triclosan Concentrations during Pregnancy and Childhood

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Abstract

Exposure to triclosan, an antimicrobial used in many consumer products, is ubiquitous in the United States, yet only limited data are available on the predictors and variability of exposure, particularly in children. We examined the patterns, variability, and predictors of urinary triclosan concentrations in 389 mother–child pairs enrolled in the Health Outcomes and Measures of the Environment Study from 2003 to 2006. We quantified triclosan in 3 urine samples collected from women between 16 weeks of pregnancy and birth and 6 urine samples collected from children between the ages of 1–8 years. For maternal and child samples, we calculated intraclass

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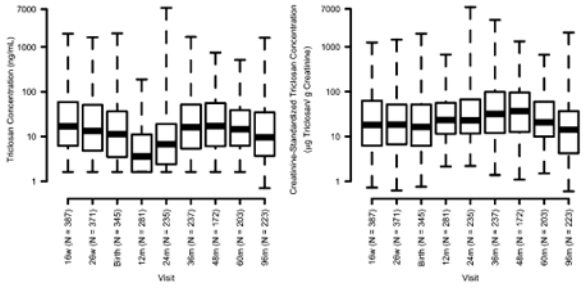
The findings and conclusions in this report are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention (CDC). Use of trade names is for identification only and does not imply endorsement by the CDC, the Public Health Service, or the US Department of Health and Human Services. The authors declare no competing financial interest.

correlation coefficients (ICCs) to assess triclosan reproducibility and identified sociodemographic predictors of triclosan. Among 8 year old children, we examined associations between triclosan and personal-care product use. We detected triclosan in >70% of urine samples. Median maternal triclosan varied across pregnancy from 17 to 11 ng/mL, while in children, median concentrations increased from 3.6 to 17 ng/mL over the first 4 years of life, declining slightly at later ages. Triclosan reproducibility was fair to good during pregnancy and for child samples taken weeks apart (ICCs = 0.4–0.6) but poor for annual child samples (ICCs = 0.2–0.4). Triclosan was 66% (95% CI: 29–113) higher in 8 year olds using hand soap compared to nonusers and increased monotonically with hand-washing frequency. Toothpaste use in children was also positively associated with triclosan. Our results suggest that urinary triclosan concentrations have modest stability over weeks to months; children are exposed to triclosan through the use of some personal-care products.

Graphical abstract



Introduction

Triclosan (5-chloro-2-(2,4-dichlorophenoxy)phenol) is an antimicrobial chemical used in a variety of consumer products, including some soaps, toothpastes, mouthwash, kitchen utensils, clothes, cleaning products, and personal-care products, such as cosmetics. The routes of exposure are primarily oral from some toothpastes and dermal from the use of some soaps or personal-care products.[1,2] Triclosan does not persist in the body and has a biological half-life of <24 h.[3] In humans, triclosan is primarily metabolized through glucuronidation of the parent compound and excreted in the urine.[2]

Because triclosan is an endocrine disruptor,[1] there is interest in characterizing the potential health effects of gestational and early-childhood exposure. Triclosan may decrease circulating thyroxine levels and interfere with thyroid hormone signaling and metabolism during gestation or childhood, adversely affecting neurobehavioral end-points sensitive to thyroid hormone disruption.[1,4,5] Prenatal urinary triclosan concentrations have also been associated with reduced weight and head circumference at birth.[6]

Prior biomonitoring studies have found detectable concentrations of triclosan in the plasma, breast milk, and urine of pregnant or lactating women from a variety of populations.[7–17] Others have also detected triclosan in the urine of infants and children (Tables S1 and S2).[18–24] However, research to characterize the variability of urinary triclosan concentrations, particularly in children, as well as to identify factors that predict triclosan

exposure is limited. The objectives of the present study were to characterize the patterns and variability of serial urinary triclosan concentrations in a cohort of mothers and their children from Cincinnati, OH and to explore associations with sociodemographic and environmental factors and, among children, personal-care product use.

Methods

Study Participants

From March 2003 to January 2006, we enrolled 468 of 1263 eligible women (37%) in the Health Outcomes and Measures of the Environment (HOME) Study, a prospective pregnancy and birth cohort in the Cincinnati, OH metropolitan area originally designed to study low-level environmental exposures and children's health. We have described participant recruitment and eligibility criteria previously.[25] We restricted the current analysis to 389 women and their singleton children who had at least one urinary triclosan measurement during pregnancy or from 1 of 6 study visits conducted between 1 and 8 years of age, respectively. The institutional review board (IRB) at Cincinnati Children's Hospital Medical Center (CCHMC) approved this study. The IRB at Brown University relinquished authority to the CCHMC IRB through an interagency agreement. The Centers for Disease Control and Prevention (CDC) IRB deferred approval to the CCHMC IRB. We obtained written informed consent from all mothers for themselves and their children.

Urinary Triclosan Concentrations

Between March 2003 and January 2006, we collected three urine samples from pregnant women at approximately 16 and 26 weeks of pregnancy and around the time of delivery. We collected urine samples from children during study visits between 2004 and 2014, which included clinic visits at age 1, 2, 3, 4, 5, and 8 years and annual home visits at age 1–3 years. We had urine samples from both the clinic and home visits for a subset of 61 children. In these instances, we used the sample collected at the clinic visit in the present analysis.

Caregivers wiped their child's genital area with a triclosan-free Wet Nap before urine collection. We collected urine onto Kendall abdominal pads placed inside the diaper for nontoilet-trained children, a training potty lined with inserts for children who were being toilet-trained, or directly into polypropylene specimen cups for children who were toilet-trained. We stored urine samples at −20 °C until shipment to the CDC, where they were stored at or below −20 °C until analysis.

We followed the recommendations of Ye et al. (2013) to minimize external contamination during urine sample collection, storage, and analysis and tested the wipes for triclosan, which was not detected.[26–28] While we have no reason to believe that triclosan was present in the inserts or cups used for urine collection, we analyzed a subset of urine samples with and without enzymatic deconjugation to determine if there were high levels of free triclosan. The results suggested that triclosan was mostly conjugated, ruling out the possibility of external contamination (data not shown). In addition, each batch of samples was analyzed with low- and high-concentration quality-control samples (coefficients of variation <10%) as well as reagent blanks. Taken together, these data do not suggest systematic contamination

during the collection or processing of the samples (additional details in the Supplemental Methods).

Using previously described analytical chemistry methods, we quantified concentrations of total (conjugated plus free) triclosan.[29] The limit of detection (LOD) was 2.3 ng/mL for all maternal samples and child samples collected at the age 1–5 year visits and 1.0 ng/mL for child samples collected at the 8 year visit. The LOD for urinary triclosan concentrations changed at age 8 years because of improvements in the triclosan assay. We assigned concentrations below the LOD a value of LOD/√2.[30] We measured urinary creatinine concentrations using a previously described assay[31] and compared different methods of accounting for urine dilution, including standardization, whereby we divided individual triclosan values by urinary creatinine (creatinine-standardized values) and adjusting for creatinine or age-specific creatinine *z*-scores as a co-variate when modeling urinary triclosan concentrations.

Predictors of Triclosan Concentrations

We examined sociodemographic and maternal factors as predictors of maternal or child urinary triclosan concentrations. Child sex and parity was obtained from medical charts, while trained research staff collected all remaining sociodemographic information (maternal and child race, household income, maternal education, and marital status) by administering standardized questionnaires to participating mothers at each study visit. Maternal depressive symptoms were measured at each visit using the Beck Depression Inventory (BDI)-II.[32] Depression may affect how a mother rates her child's behavior, which is an end point of interest for triclosan exposure. We also obtained the mothers' full scale IQ using the Wechsler Abbreviated Scale of Intelligence during postnatal visits.

We examined relations between urinary triclosan and other environmental exposures, including measures of maternal or childhood tobacco smoke, bisphenol A (BPA), and phthalate exposures. For mothers, we measured serum cotinine concentrations at 16 and 26 weeks of pregnancy and at the age 1, 2, and 3 year visits for children. We also measured BPA and five phthalate metabolites in maternal and child urine samples collected at the study visits (16 and 26 weeks for mothers, ages 1–5 and 8 years for children). The phthalates we examined were monoethyl phthalate (MEP), mono-*n*-butyl phthalate (MnBP), monoisobutyl phthalate (MiBP), mono-benzyl phthalate (MBzP), and mono-3-carboxypropyl phthalate (MCPP). We also calculated a summary measure of di-2-ethylhexyl phthalate (ΣDEHP), calculated as the molar sum of mono-2-ethylhexyl phthalate (MEHP), mono-2-ethyl-5-oxo-hexyl phthalate (MEOHP), mono-2-ethyl-5-hydroxyhexyl phthalate (MEHHP), and mono-2-ethyl-5-carboxypentyl phthalate (MECPP). We did not measure MEHP and MiBP in urine collected at the age 1, 2, and 3 year visits because we detected these analytes in the inserts used for sample collection at those ages.

During the 8 year visit, we administered a questionnaire about chemical exposures to mothers to find out about their child's use of specific personal care products (e.g., shampoo, conditioner, hand soap, toothpaste, makeup use) in the past 24 h. We also asked about the child's frequency of hand washing.

Statistical Analysis

We assessed trends in $\log_{10}$-transformed urinary triclosan concentrations for mothers (from 16 weeks gestation to birth) and children (from age 1 to 8 years), with and without creatinine standardization, using box-and-whisker plots and linear mixed models. We also calculated Pearson correlation coefficients between $\log_{10}$-transformed, maternal urinary triclosan concentrations during pregnancy, and children's urinary triclosan concentrations at each age, both unstandardized and creatinine-standardized.

We examined the reproducibility of urinary triclosan concentrations across visits using intraclass correlation coefficients (ICCs) as measures of the reproducibility of a measurement within an individual. ICCs of 0 to <0.4, 0.4 to <0.75, and ≥0.75 indicate poor, fair to good, and excellent reproducibility, respectively. We estimated between-and within-subject variability of $\log_{10}$-transformed urinary triclosan concentrations using random intercept linear mixed models with an unstructured covariance matrix. For maternal samples, we calculated ICCs between 16 and 26 weeks, 16 weeks and birth, 26 weeks and birth, and all three time points using participants with a triclosan measurement from at least two visits. For children, we calculated long-term ICCs across all six visits and for narrower age groups that included toddlers (ages 1–2 years), preschoolers (ages 3–4 years), and school-aged children (ages 5–8 years). We also calculated short-term ICCs for the subset of 61 children with both a clinic and home visit urine sample taken an average of 1.3 (range: 0.1–6.0) weeks apart at age 1–3 years.

To explore associations between urinary triclosan and sociodemographic and environmental factors, we used linear mixed models with maternal or child $\log_{10}$-transformed triclosan concentrations as the dependent variable. This approach allowed us to account for repeated measures of triclosan. We examined each sociodemographic variable individually in unadjusted models as well as in models mutually adjusted for all factors. Because the relationship between child age and urinary triclosan was not linear, models of children's triclosan concentrations were adjusted for age and age-squared terms. We calculated percent differences in maternal or child triclosan concentrations per interquartile range (IQR) increase in $\log_{10}$-transformed mean serum cotinine concentrations (averaged over 16- and 26-weeks for mothers and age 1–3 years for children) or time-varying concentrations of urinary BPA and phthalate metabolites. Maternal models were adjusted for mother's race, age, education, parity, marital status, household income, and creatinine *z*-score, while child models were adjusted for child's race, sex, age, household income, mother's education, and creatinine *z*-score.

To examine associations between children's urinary triclosan concentrations and personal care product use at the age 8-year visit, we used multivariable linear regression, adjusting for maternal education, child age, sex, race, household income, and creatinine *z*-score. Because our outcomes were $\log_{10}$-transformed, we calculated the percent difference in triclosan concentrations compared to the reference group for categorical variables or for a given increase in continuous variables by raising beta coefficients to the 10th power. We performed all analyses using R version 3.2.1 (R Foundation for Statistical Computing, Vienna, Austria).

Results

Patterns and Variability

There were 389 women with at least one urinary triclosan measurement during pregnancy or at delivery and 338 children with at least one triclosan measurement from 1 to 8 years of age. Of the participants who provided one or more urine samples, 377 (1060 repeated measures) women and 326 (1297 repeated measures) children had complete co-variate information. At the 8 year visit, 218 children provided a urine sample and had complete co-variate data, including parent-reported personal-care product use.

We detected triclosan in 84–92% of women's urine samples and 71-96% of children's samples, depending on the visit (Table S3). Median unstandardized maternal urinary triclosan concentrations decreased from 17 to 11 ng/mL between 16 weeks of pregnancy and birth (p-value for test of trend across gestational age of 0.0004; Figure 1 and Table S3). For children, the median unstandardized urinary triclosan concentrations increased from 3.6 ng/mL at 1 year to 17 ng/mL at 4 years of age, declining at later ages to approximately 10 ng/mL. We observed a significant nonlinear association between child age and urinary triclosan (p-value for child's age-squared $<10^{-16}$). We observed a similar pattern after creatinine-standardizing mother's and children's triclosan concentrations (Figure 1 and Table S3).

Maternal urinary triclosan concentrations had fair reproducibility, with ICCs ranging from 0.38 to 0.58, though the reproducibility did not increase when we examined adjacent measurements (Figure 2 and Table S4). In contrast, the ICC of children's triclosan concentrations increased with decreasing duration between measurements (Figure 2 and Table S4). Children's long-term ICCs were ≤0.28, indicating poor reproducibility of triclosan concentrations in samples collected ≥1 year apart. However, ICCs calculated over shorter timeframes (e.g., toddlers and preschoolers) had better reproducibility than ICCs calculated over longer timeframes (see Table S4). Furthermore, the short-term ICCs for the subset of 61 children with both a clinic and home visit from 1 to 3 years of age were 0.54 to 0.59 (Figure 2). The three methods used to account for urine dilution did not change the ICCs.

Average maternal triclosan concentrations during pregnancy were correlated ($p < 0.05$) with child triclosan concentrations at most ages after, but not before, creatinine standardization with Pearson correlation coefficients ranging from 0.14 to 0.38 (Table S5). These findings may be in part related to correlations between maternal and children's creatinine, which were statistically significant ($p < 0.05$) at ages 2, 3, 4, and 5 years of age (data not shown).

Demographic and Environmental Predictors

Maternal urinary triclosan concentrations during pregnancy were lower in black and unmarried women (Table 1). Compared to white women, black women had 41% (95% CI: −55, −22) lower urinary triclosan concentrations after adjustment for other sociodemographic covariates. Unmarried women had 26% (95% CI: −43, −3) lower urinary triclosan concentrations compared to married women. Urinary triclosan concentrations increased 5% (95% CI: 1, 10) per $10 000 increase in household income and also with

increasing levels of educational attainment. College graduates had 85% (95% CI: 50, 129) higher triclosan concentrations compared to women who did not graduate high school. There were no clear patterns in maternal urinary triclosan concentrations with categories of mother's age, parity, maternal BDI, or IQ scores after adjustment (see Table 1).

Black children had lower urinary triclosan concentrations compared to white children after adjustment for other sociodemographic factors (percent difference: −15%, 95% CI: −33, 7; see Table 2). Urinary triclosan concentrations in children increased slightly, 1% (95% CI: −1, 4), per $10 000 increase in household income. Children born to mothers who had at least some college education had higher urinary triclosan concentrations compared to those born to mothers with less than a high school education (Table 2). Triclosan concentrations were 27% (95% CI: −2, 66) higher for children born to mothers in the second-highest IQ category, but this relationship did not hold for those born to mothers in the highest IQ group. Child sex and maternal BDI scores were not associated with child urinary triclosan.

Maternal triclosan concentrations were 9% (95% CI: −23, 9) lower per IQR increase in mean serum cotinine. An IQR increase in the concentration of BPA and all phthalate metabolites during pregnancy was associated with greater maternal triclosan concentrations (Figure 3). Among children, triclosan concentrations were 14% (95% CI: −26, −1) lower per IQR increase in mean serum cotinine but were not associated with urinary BPA concentrations. Child triclosan concentrations were positively associated with all phthalate metabolites, with the strongest associations seen for MEP, MBzP, and MCPP (see Figure 3).

Personal-Care Product Predictors

After adjustment for co-variates, child urinary triclosan concentrations at 8 years of age were associated with parent-reported child use of toothpaste, hand soap, and bar soap in the past 24 h (Table 3). For children who had used toothpaste, triclosan concentrations were 167% (95% CI: 109, 240) higher than those who did not use toothpaste in the past 24 h. Children who had used hand soap in the past 24 h had 66% (95% CI: 29, 113) higher urinary triclosan concentrations than nonusers. Triclosan concentrations also increased monotonically with increasing frequency of parent-reported hand washing; children who washed their hands more than five times per day had 328% (95% CI: 197, 517) higher triclosan concentrations than those who washed their hands once per day or less (Figure 4). Child urinary triclosan concentrations were not associated with the use of other personal care products, such as hair products, lotion, deodorant, or mouthwash (Table 3). Child triclosan concentrations were not associated with use of makeup or specific cosmetics (e.g., mascara, lipstick, and nail polish) in the past 24 h (Table S6), although the number of children using any given product was relatively small, and makeup use was only reported for girls. The associations between urinary triclosan and toothpaste or hand soap use were strengthened after additionally adjusting for the sum of other personal care products used, while the association with bar soap was attenuated toward the null (data not shown).

Discussion

In the present study, we characterized the longitudinal patterns and variability of serial urinary triclosan concentrations in a cohort of mothers and their young children between the

second trimester of pregnancy and 8 years of age. Among pregnant women in our study, median urinary triclosan concentrations decreased across pregnancy (17 to 11 ng/mL) and were within ranges of central tendencies of urinary concentrations measured in other groups of pregnant women in the United States and Canada (7–25 ng/mL, see Table S1 for comparison across studies). [8,9,12,13,15] The three urinary triclosan concentrations obtained during pregnancy and at birth had fair to good reproducibility. Previous studies have also found that triclosan concentrations during pregnancy exhibit moderate reproducibility, reporting ICCs ranging from approximately 0.4 to 0.6.[9,11,13]

Median urinary triclosan concentrations in HOME Study children ranged from 3.6 to 17 ng/mL and were similar to, and in some cases less than, concentrations measured in comparable age groups in previous U.S. cohorts (6–32 ng/mL) [23,24,33] but somewhat higher than those measured in several European studies (≤4 ng/mL, see Table S2).[20–22] However, few prior studies have examined triclosan exposure in children age <6 years. We observed a statistically significant, nonlinear trend in urinary triclosan over childhood; concentrations increased up to 4 years of age and decreased slightly afterward. We speculate that the rise in triclosan concentrations with child's age is due to behavioral changes associated with personal care product use, as children begin to take on habits similar to their parents, which, in turn, increases their triclosan exposure. Child urinary triclosan measured in samples collected over shorter timeframes (i.e., weeks) exhibited better reproducibility compared to concentrations in samples collected one year or more apart. One previous study reported an ICC of 0.39 for triclosan, determined based on urine samples collected from 6 to 10 year old minority children over a period of 6 months.[33] Our short-term ICCs, calculated from urine samples taken an average of 1.3 weeks apart, are slightly higher than the ICC reported by Teitelbaum et al. (2008).[33]

We also investigated associations of urinary triclosan concentrations with sociodemographic factors, other environmental exposures, and personal-care product use. Maternal urinary triclosan concentrations were associated with several sociodemographic factors in our study, including maternal race, marital status, education, and household income. Black women and unmarried women had 41% and 26% lower triclosan concentrations compared to white women and married women, respectively, while concentrations increased with increasing educational attainment and household income. Previously, higher urinary triclosan concentrations have been associated with higher social class, income, and education in pregnant women from Canada and Spain[8,20] and with income in adults from the U.S. National Health and Nutrition Examination Survey.[34] In a group of pregnant women enrolled in the U.S. National Children's Study, Hispanic women had the highest urinary triclosan compared to non-Hispanic black and white women.[12] Non-Hispanic white women in our study had the highest adjusted geometric mean urinary triclosan (21 ng/mL), while women in “other” racial groups had the next highest, although the sample size in this category was only 26 women. Black children in our study also had lower triclosan concentrations in their urine compared to white children. Children's triclosan concentrations increased with increasing levels of maternal education. Differences in urinary triclosan concentrations across sociodemographic groups are likely related to personal-care product use patterns. However, triclosan concentrations did not differ between racial and ethnic groups in a multiethnic cohort of 6 to 8 year old girls from New York City; Cincinnati, OH;

and the San Francisco Bay Area.[24] As higher socioeconomic status is also typically associated with better health outcomes, future studies evaluating the potential health effects of triclosan exposure should consider adjustments for factors such as income and education to minimize potential negative confounding.[35]

In our study, triclosan concentrations were inversely associated with serum cotinine in both mothers and children. We also found positive associations between urinary triclosan and concentrations of phthalate metabolites for mothers and children; however, urinary triclosan and BPA concentrations were only associated among mothers. Consistent with our findings, urinary triclosan concentrations were lower in women who were smokers compared to women who were never smokers in a cohort of pregnant women from Canada.[8] The correlations between urinary triclosan and phthalate metabolites we observed may be partly due to shared sources of exposure because low-molecular-weight phthalates, such as diethyl phthalate, are used as solvents in some personal-care products.[36]

Among 8 year old children, urinary triclosan concentrations were associated with parent-reported child use of hand soap and toothpaste in the past 24 h, as well as with increased frequency of hand washing. Triclosan concentrations were not, however, associated with the use of other personal care products, such as hair products and cosmetics. Few previous studies have examined associations between urinary triclosan concentrations and personal care product use. Meeker et al. (2013) found that increased urinary triclosan concentrations were associated with recent liquid soap and hairspray use, although this analysis was done in Puerto Rican pregnant women, not children.[11] Children from the Study of Use of Products and Exposure-Related Behavior that used Colgate toothpaste had 87% higher urinary triclosan concentrations compared to users of other toothpaste brands.[23] In a cohort of Swedish mothers and their children, no notable differences in triclosan concentrations were detected between users and nonusers of products possibly containing triclosan, such as mouthwash and hand disinfectants, but the study also had a low percentage (~36%) of urine samples with detectable triclosan concentrations.[22] Given the recent U.S. Food and Drug Administration's regulation banning the use of triclosan in over-the-counter hand soaps,[37] future biomonitoring studies will be instrumental to determine if this regulation reduces triclosan exposure in the United States.

Our study has several strengths. We measured urinary triclosan concentrations longitudinally among mothers during the latter two-thirds of pregnancy and among children from infancy to the time that they were in elementary school. This time period encompasses several potentially sensitive windows of heightened vulnerability to triclosan, which has been found to decrease circulating thyroxine levels and interfere with thyroid hormone signaling and metabolism during gestation and childhood.[4,5,38] In addition, at 8 years, we administered to the mothers an exposure questionnaire to collect detailed information on their child's personal care product use in the past 24 h, including hand soap, toothpaste, and other products that may contain triclosan. However, only 3% of children in our study did not use toothpaste, and we did not ask about use of specific product brands; thus, we are unable to distinguish between, for example, children using triclosan-containing brands of toothpaste and those using triclosan-free brands.

There is the possibility for misclassification of urinary triclosan concentrations, particularly among children, where we had one urine sample available to represent exposure over periods of 1 year or more. In fact triclosan concentrations were more reproducible in urine samples collected weeks apart compared to samples collected years apart. Nevertheless, as indicated by the short-term ICCs in our study, triclosan concentrations have better reproducibility compared to other nonpersistent chemicals, such as bisphenol A.[39] Our analysis of short-term ICCs suggests that triclosan exposure may be relatively stable over a period of a few weeks, and, thus, a single sample may be adequate to assess exposure at distinct and defined life-stages. Future epidemiology studies might consider collecting, and possibly pooling, multiple urine samples over a period of weeks or months at each age from pregnant women or children to minimize exposure misclassification for that life-stage.[40–42]

While individual patterns of personal care product use are likely similar day-to-day, events that disrupt typical routines (e.g., travel) could affect triclosan urinary concentrations and increase within-person variation. There is also the potential for misclassification of personal care product use because parents reported this information on behalf of their child. Thus, the parent's answers may not reflect the child's actual product use, particularly outside the home. Further, it is possible there are other potential sources of triclosan exposure in young children that we were unable to investigate; for example, triclosan is an ingredient in some clothing fabrics (Microban), which we did not ask about in our exposure questionnaire.

The use of creatinine to adjust for urine dilution is another limitation. Creatinine adjustment may not account for changes in creatinine metabolism and excretion that occur during pregnancy or over childhood. We addressed this by adjusting for age-specific child creatinine *z*-scores in our regression models as an alternative to using the creatinine-standardized triclosan concentrations, but there could still be additional misclassification of triclosan exposure, especially for women or children with dilute or concentrated urine. Finally, the relatively small number of children who engaged in less-common behaviors, such as hair treatments and makeup use, may have precluded our ability to detect associations between urinary triclosan concentrations and some sources of exposure.

In conclusion, we found widespread triclosan exposure in a cohort of pregnant women and their children. Triclosan measured in serial urine samples collected over a relatively short time frame of 2 weeks show good reproducibility and may adequately capture triclosan exposure within defined periods. Sociodemographic factors such as race, income, and education should be considered as possible negative confounding factors in future work because they may be associated with both higher urinary triclosan concentrations and better health outcomes. Finally, we found that the use of hand soap and toothpaste was positively associated with urinary triclosan concentrations in school-aged children; thus, triclosan exposure may be modifiable through behavioral or regulatory interventions.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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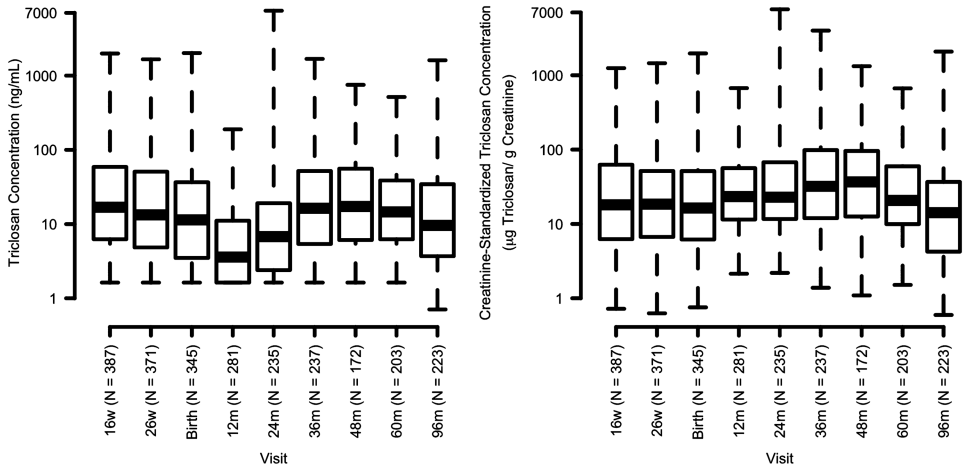

Figure 1.

Box-and-whisker plots of unstandardized (ng/mL) and creatinine-standardized (μg triclosan/g creatinine) urinary triclosan concentrations in HOME Study mothers and children.

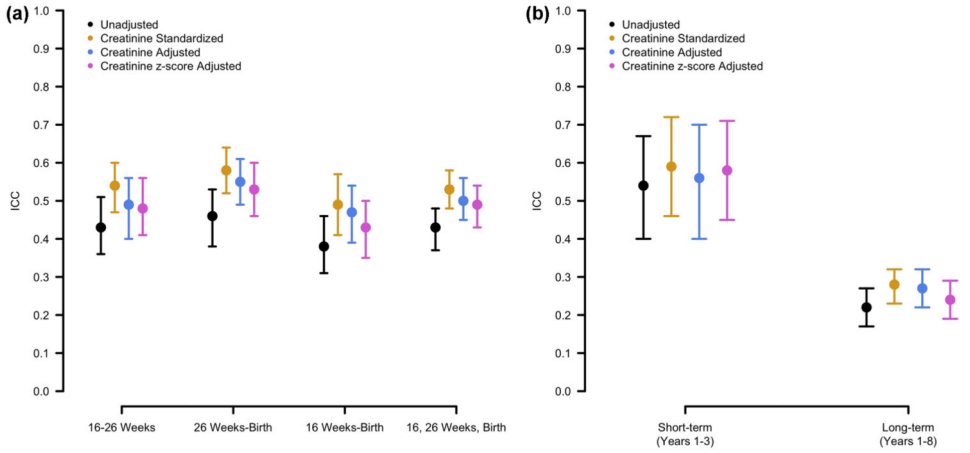

Figure 2.

Intraclass correlation coefficients (ICCs) of HOME Study (A) maternal and (B) child urinary triclosan concentrations.

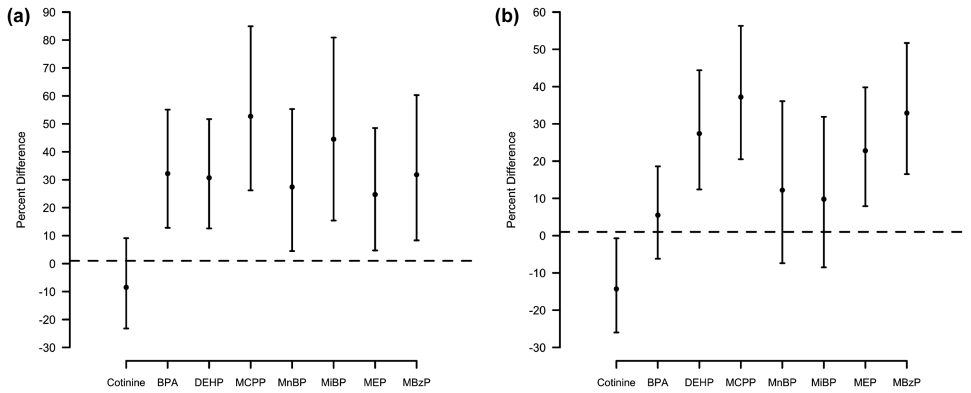

Figure 3.

Percent change (with 95% confidence interval) in HOME Study (A) mothers' and (B) children's urinary triclosan concentrations per interquartile range increase in serum cotinine, urinary BPA, and urinary phthalate metabolite concentrations. For mothers, measurements at 16 and 26 weeks were used; for children, measurements at the 1–5 and 8 year visits were used except for mean serum cotinine, which was only measured at ages 1–3 for children. Urinary phthalate metabolites included MCPP, MnBP, MiBP, MEP, MBzP, and the sum of DEHP metabolites (MEHP + MEHHP + MEOHP + MECPP). Models in panel A were adjusted for mother's race, age, education, parity, marital status, household income, and creatinine *z*-score. Models in panel B were adjusted for child's race, sex, age, household income, mother's education, and creatinine *z*-score.

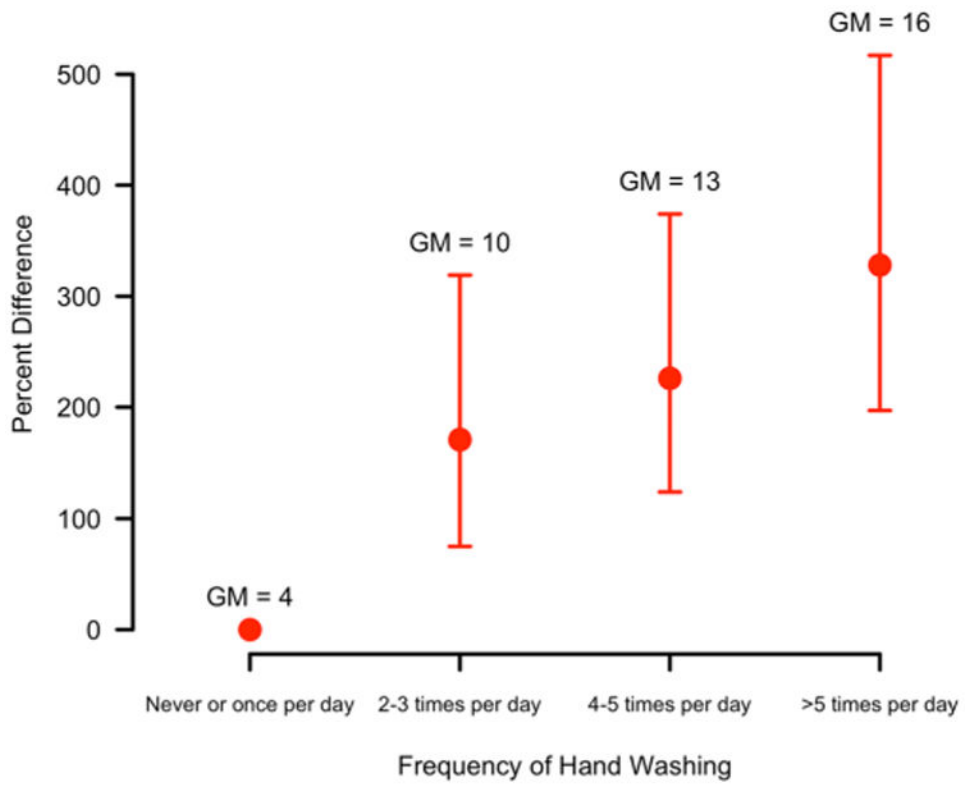

Figure 4.

Adjusted percent difference in the HOME Study children's urinary triclosan concentrations at 8 years of age according to parent-reported child frequency of hand washing. Adjusted for child's race, sex, age, household income, mother's education, and creatinine *z*-score.

Table 1

Geometric Mean (GM) and Percent Difference (% diff) in HOME Study Mothers' Urinary Triclosan Concentrations at 16 Weeks, 26 Weeks, and Birth, According to Sociodemographic and Maternal Mental Health Factors (*N* = 375 with a Total of 1055 Repeated Measures)

variable	N (%)	number of measurements	unadjusted GM (ng/mL)	unadjusted % diff (95% CI)[a]	adjusted GM (ng/mL)	adjusted % diff (95% CI)[b]
maternal race						
non-hispanic white	235 (62.3)	673	22	ref	18	ref
non-hispanic black	114 (30.4)	311	9	−57 (−66, −46)	11	−41 (−55, −22)
other	26 (6.9)	71	15	−32 (−58, 10)	13	−27 (−56, 21)
marital status						
married	244 (65.1)	699	21	ref	17	ref
unmarried	131 (34.9)	356	10	−52 (−62, −40)	12	−26 (−43, −3)
mother's age (years)						
25-34	209 (55.4)	596	19	ref	16	ref
<25	89 (23.6)	240	11	−42 (−56, −24)	14	−12 (−35, 20)
>34	79 (21.0)	224	19	1 (−24, 34)	13	−15 (−39, 17)
parity						
0	169 (45.1)	469	18	ref	15	ref
≥1	206 (54.9)	586	15	−17 (−30, −0.3)	14	−8 (−24, 13)
maternal BDI-II						
minimal: < 14	292 (77.9)	829	19	ref	15	ref
mild: 14–19	52 (13.9)	141	12	−36 (−55, −9)	14	−12 (−39, 26)
moderate and severe: > 19	31 (8.3)	85	10	−48 (−67, −18)	12	−22 (−51, 24)
maternal IQ						
58–101	89 (39.4)	244	11	ref	13	ref
102–114	65 (28.8)	191	23	111 (57, 184)	22	68 (15, 146)
115+	72 (31.9)	203	20	82 (37, 142)	16	27 (−17, 92)
maternal education						
less than grade 12	39 (10.4)	105	8	ref.	11	ref
high school graduate	52 (13.9)	138	12	40 (−2, 98)	14	27 (−12, 84)
some college	94 (25.1)	266	14	61 (25, 109)	15	38 (6, 81)
college graduate	190 (50.7)	546	23	171 (126, 225)	20	85 (50, 129)

variable	N (%)	number of measurements	unadjusted GM (ng/mL)	unadjusted % diff (95% CI)[a]	adjusted GM (ng/mL)	adjusted % diff (95% CI)[b]
household income						
per $10 000 increase	375	1,055	–	9 (5, 12)	–	5 (1, 10)

[a] Adjusted for creatinine *z*-score and gestational week.

[b] Adjusted for creatinine *z*-score, gestational week, and all covariates presented in the table (household income and maternal education are time-varying).

Table 2

Geometric Mean (GM) and Percent Difference (% diff) in HOME Study Children's Urinary Triclosan Concentrations at 1–8 Years of Age, According to Sociodemographic and Household Factors (*N* = 314 with a Total of 1254 Repeated Measures)

variable	N (%)	number of measurements	unadjusted GM (ng/mL)	unadjusted % diff (95% CI)[a]	adjusted GM (ng/mL)	adjusted % diff (95% CI)[b]
child race						
non-hispanic white	199 (63.4)	823	12	ref	10	ref
non-hispanic black	94 (29.9)	349	8	−34 (−46, −20)	8	−15 (−33, 7)
other	21 (6.7)	82	15	21 (−20, 82)	13	34 (−12, 104)
child sex						
female	173 (55.1)	686	11	ref	9	ref
male	141 (44.9)	568	11	0.5 (−15, 18)	9	−1 (−19, 19)
household income						
per $10 000 increase	314	1254	–	3 (1, 5)	–	1 (−1, 4)
maternal education						
less than grade 12	27 (8.6)	97	8	ref	8	ref
high school graduate	37 (11.8)	129	6	−22 (−42, 6)	6	−23 (−44, 6)
some college	82 (26.1)	350	11	44 (18, 74)	12	39 (13, 71)
college graduate	168 (53.5)	678	13	61 (40, 86)	12	47 (25, 72)
child age						
age	314	1254	–	114 (90, 140)	–	115 (91, 141)
age-squared	314	1254		−7 (−8, −6)		−7 (−8, −6)
maternal BDI-II						
minimal: < 14	255 (81.2)	1039	11	ref	9	ref
mild: 14–19	35 (11.1)	120	10	−14 (−39, 21)	10	6 (-25, 49)
moderate and severe: > 19	24 (7.6)	95	10	−14 (−42, 27)	10	−13 (−24, 68)
maternal IQ						
58–101	177 (56.4)	596	9	ref	9	ref
102–114	65 (20.7)	302	14	48 (18, 86)	11	27 (−2, 66)
115+	72 (22.9)	356	12	26 (2, 56)	9	4 (−20, 36)

[a] Adjusted for creatinine *z*-score, age squared, and child age squared.

[b] Adjusted for creatinine *z*-score, child age, and child age squared and all covariates presented in the table (household income and maternal education are time-varying).

Table 3

Unadjusted and Adjusted Percent Difference (% diff) in HOME Study Children's Urinary Triclosan Concentrations at 8 Years of Age According to Parent-Reported Child Use of Specific Personal Care Products in the Past 24 h (*N* = 214)[a]

product	used or not	*N* (%)	unadjusted GM (ng/mL)	unadjusted % diff (95% CI)	adjusted GM (ng/mL)	adjusted % diff (95% CI)
toothpaste	not used	6 (3%)	4.1	ref	4.6	ref
	used	208 (97%)	12.4	204 (146, 275)	12.4	167 (109, 240)
hand soap	not used	29 (14%)	6.9	ref	7.7	ref
	used	185 (86%)	13.1	91 (53, 138)	12.9	66 (29, 113)
bar soap	not used	105 (49%)	11.3	ref	10.3	ref
	used	109 (51%)	12.6	11 (−17, 49)	14.3	40 (1, 93)
hand sanitizer	not used	116 (54%)	11.6	ref	12.0	ref
	used	98 (46%)	12.4	6 (−22, 45)	12.3	3 (−27, 43)
mouthwash	not used	166 (78%)	11.9	ref	11.9	ref
	used	48 (22%)	12.1	2 (−34, 59)	12.9	9 (−31, 71)
shampoo	not used	107 (50%)	10.9	ref	12.0	ref
	used	107 (50%)	13.1	20 (−11, 61)	12.2	1 (−27, 40)
lotion or sunscreen	not used	120 (56%)	12.4	ref	11.9	ref
	used	94 (44%)	11.5	−7 (−32, 27)	12.3	4 (−26, 46)
liquid soap or body wash	not used	93 (43%)	12.1	ref	12.7	ref
	used	121 (57%)	11.9	−2 (−26, 30)	11.7	−8 (−32, 25)
deodorant	not used	169 (79%)	12.9	ref	12.3	ref
	used	45 (21%)	9.1	−29 (−55, 11)	11.3	−9 (−45, 52)
hair treatments	not used	212 (99%)	12.0	ref	12.1	ref
	used	2 (1%)	10.9	−9 (−90, 682)	10.4	−14 (−90, 636)
conditioner	not used	148 (69%)	12.2	ref	12.9	ref
	used	66 (31%)	11.6	−5 (−34, 39)	10.4	−19 (−47, 22)
hair spray or gel	not used	180 (84%)	12.7	ref	12.7	ref
	used	34 (16%)	8.9	−30 (−58, 18)	9.2	−28 (−59, 27)

[a]Adjusted for race, income, education, sex, and creatinine.